

Patient Presentation

- CC: " I feel lightheaded and have dark bowel movements".
- HPI.
 - David Marcus is a 74 yo man with a 24-hour history of dizziness, nausea, and dark stool. He stated that he has a hx of PUD dating back to late 1960s, and a mild UGI bleed several years ago. In the ED, he was not orthostatic; An NG tube was placed, his stool was guaiac tested, blood was drawn, an ECG was performed, and an IV was started for crystalloid administration

PMH

- PUD history since 1968
- UGI bleed in 1989 with gastritis
- Refractory RA x 6years with multiple failed treatments
- HTN x 10 years
- FH
- Younger brother also has PUD: otherwise non-contributory
- SH
- Retired Vet, non-smoker, moderate social alcohol use; married

MEDs

- Piroxicam 20mg po QD for two months
- Atenolol 50mg QD x 9 years
- ALLergies
- NKDA
- ROS not remarkable
- Some nausea
- PE
- LA (S) 151/70 P. 90
- LA (ST) 145/64 P.110
- RR16 T37.6; Wt 88KG Ht 173cm
- HEENT: PERRLA;EOMI; discs
- Flat; no AV nicking,

Neuro and Labs

- A&O x 3; CN 2-12 grossly intact
- Na 142mEq/L K+ 4.1
- Chl 102 CO2 22 BUN 30; Serum Creatinine 1.1 glucose 124
- Hgb 11.9 Hematocrit 36.1
- Platelets 299,000/mm3
- WBC 7500/mm3
- ECG NSR
- EGD- Pre-pyloric, distal gastric ulcer with a blood clot

Problem Identification

- What are the risk factors for this patient?
- Elderly
- Hx of PUD
- Underlying CD
- Recurrent GI prob(s)
- NSAIDS
- Acute and long term MGT goals?
- Relieve pain
- Stop relapses
- Heal the ulcer
- Avoid complications
- Stop or reduce NSAIDS

Acute and LT MGT CONT.

- If NSAID stopped, full Dose Rx with either H2RAs, PPI, or sucralfate will heal most ulcers
- Monitor pt. for relief of ulcer pain as well as potential adverse drug effects & drug interaction
- Therapeutic alternatives?
- Medications
- Surgery
- Pharmacotherapeutic
- Alternatives?
- Drugs with short half-lives

Optimal Plan

- Stop the bleeding
- Relieve the pain
- Stop relapses
- Avoid Complications
- Educate the patient
- Treatment measures in the ED?
- Control acid secretion and treat the type of bleeding
- A. Oral antacids to be given @ freq. Intervals
- May be hourly

Pharmacotherapeutic plan to heal ulcer

- If the NSAID is continued, treatment with a conventional H2RA or PPI anti-ulcer regimen will promote healing, but ulcers tend to heal @ a slower rate. A longer duration of RX is indicated.
- Alternatively higher doses of a PPI (e.g. omeprazole 40mg/d may be preferred in pts with GU on NSAIDS
- Sucralfate will heal most ulcers

Assessment Parameters?

- Success of Therapy
- Parameter monitors
- A. Urea breath tests
- B. Serum Antibody Titers
- C. Repeat Endoscopy & biopsy
- D. If confirmation indicated, none of above tests should be done
- Patients who remain symptomatic after treatment, should be referred to a specialist for management.

Patient Counseling @ Discharge?

- Continue no smoking regimen
- Stop drinking or no more than one drink per week
- Take med with a small amt of food, milk, or antacid to minimize stomach upset
- Don't recline for 15-30 mins after taking the med
- Report symptoms of GI bleed
- Tarry stool
- Orthostasis
- Allergy
- Fluid retention

Follow up?

- Long term anti-ulcer prophylaxis (Yes)
- Misoprostol (cytotec) is the only drug that is FDA approved for prevention of NSAID induced ulcers
- Dose: 200mg QID
- Diarrhea + abd. Cramps may limit pat. compliance
- Start therapy with lower doses & advance to therapeutic levels to limit GI adverse effects which are usually transient
- Patient Info?
- Educate about possible
- Adverse effects

Synthetic PGE Therapy

- Was this patient a candidate?
- Yes
- Co-admin of misoprostol 200mg QID with NSAIDS is indicated for high risk patients.
- Complications
 - Erosions
 - Ulcerations
 - Agents less likely
 - PGE synthetics
 - Prophylaxis
 - PGE synthetics vs anti-secretory agents
 - Contraindication for PGE

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- Learning Objectives:
- Understand the significance and etiology of NSAID-induced gastropathy
- Determine which patients should be considered for primary prophylaxis of NSAID-GI
- Select appropriate RX for RX and prevention of NSAID GI
- Recognize MonitoringParameters used in evaluating prophylaxis

Pathogenesis

- The 2nd most common cause of peptic ulcer formation
- Damage to the mucosa
- Caused by use of NSAIDS
- Two mechanisms:
- NSAIDS damage the mucosa thru direct action
- A systemic effect occurs whereby endogenous prostaglandin synthesis is inhibited

Pathogenesis Cont...

- Historically, PUD has affected males and females in a 2:1 ratio
- Recently, the occurrence has declined in young men and increased in women
- NSAID related ulcers are more likely to be gastric than duodenal
- The elderly and patients with NSAID-induced ulcers are often symptom free until
- Bleeding/perforation
